An outbreak of foodborne botulism in Ontario

Mona R Loutfy MD, FRCPC[1], John W Austin PhD[2], Burke Blanchfield[2], Ignatius W Fong MD FRCPC[1]

MR Loutfy, JW Austin, B Blanchfield, IW Fong. An outbreak of foodborne botulism in Ontario. Can J Infect Dis 2003;14(4):206-209.

Botulism is a rare paralytic illness resulting from a potent neurotoxin produced by *Clostridium botulinum*. Botulism in Canada is predominately due to *C botulinum* type E and affects mainly the First Nations and Inuit populations. The most recent outbreak of botulism in Ontario was in Ottawa in 1991 and was caused by *C botulinum* type A. We report an outbreak of foodborne type B botulism in Ontario, which implicated home-canned tomatoes. The outbreak was characterized by mild symptoms in two cases and moderately severe illness in one case. The investigation shows the importance of considering the diagnosis of botulism in patients presenting with cranial nerve and autonomic dysfunction, especially when combined with gastrointestinal complaints; it also highlights the importance of proper home canning technique.

Key words: *Botulism; Clostridium botulinum; Ontario; Outbreak*

Une flambée de botulisme d'origine alimentaire en Ontario

Le botulisme est une maladie paralytique rare causée par une neurotoxine puissante produite par le *Clostridium botulinum*. Au Canada, le botulisme est surtout attribuable à au *C botulinum* de type E, et il touche surtout les populations inuites et des Premières nations. La plus récente flambée de botulisme en Ontario a eu lieu à Ottawa, en 1991, et a été causée par le *C botulinum* de type A. Nous déclarons une flambée de botulisme d'origine alimentaire de type B en Ontario, causée par des tomates mises en conserve à la maison. La flambée a été caractérisée par des symptômes bénins dans deux cas, et par une maladie modérée dans un cas. L'exploration révèle l'importance d'envisager un diagnostic de botulisme chez les patients présentant une dysfonction du nerf crânien et du système nerveux autonome, surtout lorsque celle-ci s'associe à des troubles gastro-intestinaux. Elle fait également état de l'importance d'utiliser des techniques convenables de mise en conserve.

Botulism is a neuroparalytic illness resulting from a potent toxin produced by the bacterium *Clostridium botulinum* (1). Foodborne botulism results from the ingestion of foods contaminated with one of three preformed toxins (A, B or E). Although foodborne botulism is a rare disease, its manifestations can be severe and can progress rapidly. Because of the potential severity of the disease and the possibility of exposure of a large number of people to contaminated products, foodborne botulism is a public health emergency requiring rapid investigation. Hauschild and Gauvreau published a thorough summary of all the botulism outbreaks in Canada between 1971 and 1984 (2). There were 61 outbreaks with 122 cases and 22 deaths. The Botulism Reference Service for Canada also summarized the botulism outbreaks in Canada between 1976 and 1987, in the year 1989, and between 1995 and 1997 (3-17). In the three most recent reports, in 1995, 1996 and 1997, there were five to seven outbreaks per year with 13, 10 and 18 cases, respectively (15-17). All of the outbreaks have been in Quebec, North West Territories, Yukon, British Columbia and Nunavut, and have primarily affected the First Nations population, particularly the Inuit. Fifty-nine per cent of the outbreaks were due to contaminated mammal meat (seal, whale, walrus) and 23% were due to fermented salmon eggs or fish. Only three (16%) outbreaks were due to home-canned foods and one (5%) was due to a commercial product. The majority (90%) of outbreaks in Canada are due to serotype E. The most recent outbreak of botulism in Ontario was in Ottawa in 1991 and was due to serotype A; however, the outbreak was never reported (KL Dodds, personal communication). Three cases were involved in this outbreak, which was caused by home-canned asparagus. The last reported case of botulism in Ontario was in 1972 (2). It was a case of type B botulism related to contaminated home-salted pork.

This report summarizes the investigation and outcome of an outbreak of foodborne type B botulism in Ontario that implicated home-canned tomatoes. The outbreak was characterized by relatively mild symptoms in two cases and moderately severe illness in one case and was caused by an unlikely food vehicle. The investigation shows the importance of considering the diagnosis of botulism soon after patients present with acute cranial nerve dysfunction, autonomic dysfunction and gastrointestinal complaints; it also highlights the importance of proper home canning techniques.

THE OUTBREAK

On January 11, 1999, a 56-year-old Italian-born man (case 1) visited a walk-in clinic in Woodbridge, Ontario. He complained of heartburn, vomiting and bloating, and was sent to the nearest emergency department, at another hospital. An abdominal radiograph revealed dilated loops of small bowel with air-fluid levels. A nasogastric tube was inserted and the patient was taken to the operating room with a presumptive diagnosis of volvulus. At laparotomy, the small bowel was dilated to the mid-jejenum and collapsed distally with no

[1]*Department of Medicine, University of Toronto, Toronto, Ontario;* [2]*Botulism Reference Service for Canada, Health Protection Branch, Health Canada, Ottawa, Ontario and Division of Infectious Disease, St Michael's Hospital, Toronto, Ontario*
Correspondence: Dr Ignatius W Fong, Division of Infectious Diseases, St Michael's Hospital, 30 Bond Street, Toronto, Ontario M5B 1W8. Telephone 905-829-4770 ext 127, fax 905-829-4799, e-mail fongi@smh.toronto.on.ca
Received for publication December 23, 2002. Accepted April 29, 2003

 ©2003 Pulsus Group Inc. All rights reserved

lesion. Six days later, he developed dysphagia, ptosis, blurred vision and diplopia. After recovery from the intestinal ileus, the patient required a gastrojejenostomy tube for feeding because of severe dysphagia. The patient was discharged after several weeks of hospitalization.

On the same day in January, case 1's wife (case 2) developed heartburn, abdominal discomfort and diarrhea. The next day, she visited her family doctor and complained of dry mouth, dysphagia and generalized weakness. Her symptoms worsened over the next week and she was subsequently assessed at the St Michael's Hospital emergency department.

On physical examination, case 2 looked ill, her pupils were fixed and dilated, and ptosis was present. Her mouth was severely dry and she had a diminished gag reflex. She had significant postural hypotension with a supine heart rate of 84 beats per minute and a blood pressure of 100/70 mmHg; on standing her heart rate was 88 beats per minute and her blood pressure decreased to 60/30 mmHg. Neurological examination revealed diminished power (4/5) of the hip flexors and small muscles of the hands bilaterally.

Their son-in-law (case 3) developed abdominal pain and vomiting after a large family meal on January 10, 1999. Two days later he developed severe constipation, bloating, blurred vision, ptosis, diplopia and dry mouth. He visited his family doctor who diagnosed a viral illness. His symptoms persisted and he was assessed in the emergency department at St Michael's Hospital 10 days after the onset of symptoms. A physical examination of case 3 revealed an otherwise healthy young man sipping water constantly from a bottle. His pupils were fixed and dilated and ptosis was present. His mouth was severely dry and his gag reflex was diminished. He also had postural hypotension with a supine heart rate of 84 beats per minute and blood pressure of 110/78 mmHg; on standing, his heart rate was 92 beats per minute and his blood pressure decreased to 84/60 mmHg. The rest of his neurological examination was normal.

A clinical diagnosis of botulism was made in the three patients. Stool and blood samples from cases 2 and 3 were sent for botulinum toxin tests. Stool samples were also sent for *C botulinum* culture. Case 1's attending physician was notified of the diagnosis of botulism in the family and the standard dose of Botulism Antitoxin Trivalent (Equine) Types A, B and E (Aventis Pasteur, Canada) was administered because of the severity of his illness. Cases 2 and 3 were treated symptomatically and all three patients had complete resolution of their symptoms by three months. It was determined that the three patients had eaten together on January 5, 9 and 10, 1999.

METHODS

Clinical and epidemiological investigation

Hypothesis-generating interviews and food histories were carried out with the three patients and all available persons present at the three meals. Each step in the preparation and storage of foods was reviewed. All persons present at the meals except one were questioned regarding symptomology and examined for signs consistent with botulism.

Laboratory investigation

Stool and serum samples were obtained from those interviewed and were assayed for botulinum toxin. Stool specimens were cultured for *C botulinum* as described elsewhere (18). Toxin was serotyped and measured in mouse minimum lethal doses using the mouse bioassay (19).

Samples of potentially contaminated foods from the family's home were assayed for botulinum toxin and were cultured for *C botulinum*. The pH of the foods was determined to identify which food would have the proper conditions to allow for *C botulinum* growth.

RESULTS

Epidemiological findings

Eight people were at risk of exposure to the botulinum toxin-containing food: an Italian family consisting of the father (case 1), the mother (case 2), three daughters, a son-in-law (case 3), a boyfriend and a priest visiting from Italy.

The family ate three meals together in the week preceding the outbreak (one on January 5, 1999, one on Jaunuary 9 and one on January 10). The meal on January 10, 1999 is the one that most likely contained the contaminated food because symptoms usually develop within 24 hours. On January 5, the family ate commercially prepared marinated seafood antipasto, bocconcini cheese, bruschetta, olives and pasta with tomato sauce made from home-canned tomatoes. On January 9, the family ate vegetable soup, cheese, pork, oyster mushrooms in oil and commercially prepared marinated seafood antipasto. On Sunday, January 10, they had a large family meal and invited a priest from Italy and the boyfriend of one of the daughters. The meal consisted of bocconcini cheese, prosciutto, olives, bruschetta, pasta with tomato sauce made from home-canned tomatoes kept in the son-in-law's (case 3) cellar and a fish dish with five kinds of fresh fish in tomato sauce made from home-canned tomatoes from the mother's (case #2) cellar.

After taking detailed food histories from each person, it was concluded that the pasta's tomato sauce and the fish in tomato sauce made on January 10 were the likely causal foods. Table 1 summarizes the clinical features, dishes eaten by each individual and the diagnostic results.

Clinical findings and laboratory confirmation of botulism

The three affected patients' clinical findings were described above. The three daughters and the boyfriend were asymptomatic and had a negative physical examination. The priest had returned to Italy – when contacted by telephone he revealed that he was asymptomatic.

Laboratory studies confirmed that botulism was likely responsible for the outbreak. *C botulinum* type B was isolated from a stool sample from case 3's wife. Although she was asymptomatic, *C botulinum* should not be recovered in normal stool. Stool cultures from the remaining five persons tested were negative. Botulinum toxin was not detected in any stool or serum sample. No stool sample from case 1 was sent for examination because of obstipation secondary to paralytic ileus.

Preparation and laboratory investigation of food

Case 3's wife made the tomato sauce for the pasta from home-canned tomatoes that were stored in their cellar. The temperature of the cellar was checked regularly and was kept below 4°C all year round. Her mother (case 2) made the tomato sauce for the fish from home-canned tomatoes that were stored in her cellar. The temperature of this cellar was warmer than 6°C when it was checked during the investigation. The entire family usually carried out the tomato canning annually.

TABLE 1
Botulism outbreak and relationship to a family dinner

Subjects	Dishes consumed	Symptoms/signs	Hospitalization	Results of diagnostic tests for *C botulinum*
1. Father (Case #1)	All dishes	Heartburn, vomiting, bloating, ileus, dysphagia, ptosis, diplopia, blurred vision, obstipation	Several weeks	No stool obtained, serum negative
2. Mother (Case #2)	All dishes except marinated seafood antipasto	Heart burn, abdominal discomfort, diarrhea, dry mouth dysphagia, weakness, fixed dilated pupils, ptosis, decreased gag reflex, postural hypotension	Managed as outpatient	Stool and serum negative
3. Son-in-law (Case #3)	All dishes	Vomiting, abdominal pain, constipation, bloating, blurred vision, dry mouth, fixed dilated pupils, ptosis, decreased gag reflex, postural hypotension	Managed as outpatient	Stool and serum negative
4. 1st Daughter (Wife of Case #3)	All dishes except marinated seafood antipasto	None	Seen in clinic	Stool positive, serum not tested
5. 2nd Daughter	All dishes except fish and seafood antipasto	None	Seen in clinic	Stool negative, serum not tested
6. 3rd Daughter	All dishes except fish and seafood antipasto	None	Seen in clinic	Stool negative, serum not tested
7. Boyfriend	Pasta only	None	Seen in clinic	Stool negative, serum not obtained
8. Priest	All dishes	None	Not seen	None obtained

Tomatoes were purchased from a farm in southwestern Ontario, boiled for 1 h, then placed in glass jars without added salt or acidifying agent. The seafood antipasto was kept refrigerated after opening.

The tomato paste and tomato sauce used for the pasta and the seafood antipasto were sent to the Botulism Reference Service for analysis. None of the fish with tomato sauce was left for examination. The tomato paste, tomato sauce and seafood antipasto were negative for neurotoxin and viable *C botulinum*, and had a pH of 4.40, 4.48 and 4.93, respectively. A jar of home-canned tomatoes with a pH of 4.55 was also negative for neurotoxin and viable *C botulinum*.

DISCUSSION

A confirmed outbreak of foodborne botulism type B occurred in Ontario in January 1999. After an epidemiological investigation, the most likely causal food was determined to be home-canned tomatoes that were kept in a cellar that was warmer than 6°C. However, the suspect food was not confirmed as contaminated because the suspected dish (fish in tomato sauce) was not available for testing.

Most outbreaks of botulism result from eating improperly preserved home-canned foods, especially vegetables (asparagus, green beans and peppers) and fish (20,21). Although heating at 100°C for 10 minutes can destroy botulinum toxin in food, the spores are heat-resistant and can survive prolonged heating. To destroy *C botulinum* spores, food must be heated under pressure to temperatures substantially greater than 100°C. Certain environmental conditions, such as the absence of oxygen (anaerobic conditions), a pH of more than 4.6, warm temperatures (greater than 4°C), moisture content (water activity) and a lack of competing bacterial flora promote the production of botulinum toxin in foods contaminated with *C botulinum* (22). The process used to produce the canned tomatoes epidemiologically implicated in the outbreak probably failed to inactivate *C botulinum* spores and may have provided conditions suitable for growth. Because of their acidic nature, tomatoes are an uncommon food to cause botulism. To improve their taste, however, some varieties of tomatoes are bred to have low acidity. This alteration may cause the pH to be just high enough to allow for the growth of *C botulinum* and the production of its toxin. In the present outbreak, the tomatoes were the likely original source of spores. *C botulinum* is a common soil organism in Canada and the spores are common on the surfaces of raw vegetables (23-25). The relatively low acidity (pH 4.55) of the home-canned tomatoes and the high storage temperature may have allowed spore germination and toxin production (26). It has also been reported that the growth of molds may result in a higher pH, allowing *C botulinum* to grow (26).

The Food and Drug Administration recommends the addition of antimicrobial growth inhibitors or acidifying agents to canned vegetables to prevent *C botulinum* spore germination and toxin production (19,22). The acidifying agents can be either citric acid or lemon juice, both of which keep the pH at less than 4.6. In addition, the jars should be kept cooler than 4°C, which also prevents the growth of *C botulinum* spores. Boiling food for 10 minutes before eating will also destroy botulinum neurotoxin.

Because of the delay in clinical diagnosis in the present outbreak, it is not surprising that the laboratory studies could not confirm the diagnoses of botulism in most of the cases. The severity of the disease was mild to moderate with one patient carrying *C botulinum* being asymptomatic. The severity of botulism correlates with the amount of botulinum toxin ingested (27). The range of severity of illness seen in this outbreak may have resulted from the unequal distribution of toxin in the tomato sauce or from the partial inactivation of the toxin by heat. However, it was surprising that the priest was asymptomatic, because he ate twice as much of the contaminated food as the others. People have different susceptibilities to botulism toxin and the priest may have been partially immune (15). This outbreak highlights both the importance of proper home-canning technique and the importance of early recognition of a rare disease. People who prepare canned vegetables at home

should be aware that this practice is hazardous, especially if such foods are allowed to remain above 'refrigerator temperature' (warmer than 4°C) (22). Botulism should be considered early in the differential diagnosis of patients presenting with descending paralysis, ptosis, extraocular palsies and autonomic dysfunction, especially when combined with gastrointestinal symptoms. A detailed food history and examination of potentially contaminated leftover food are important aspects of the investigation of a suspected botulism outbreak. If botulism is a concern, the Botulism Reference Service in Ottawa should be contacted to aid in the investigation. The administration of Botulism Antitoxin Tirvalent (Equine) Types A, B, and E (Aventis Pasteur, Canada) should be considered in all cases and has been shown to reduce the degree of paralysis and rate of mortality, especially when administered early after the onset of symptoms (28).

ACKNOWLEDGEMENTS: The authors thank Dawn Bajhan and Debbie Reid for their secretarial and organizational support.

REFERENCES

1. Shapiro RL, Hatheway C, Swerdlow DL. Botulism in the United States: A clinical and epidemiologic review. Ann Intern Med 1998;129:221-8.
2. Hauschild AH, Gauvreau L. Food-borne botulism in Canada, 1971-84. CMAJ 1985;133:1141-6.
3. Hauschild A. Botulism in Canada – Summary for 1976. Can Dis W Rep 1977;3:46-7.
4. Hauschild A, Gauvreau L, Bowmer EJ. Botulism in Canada – Summary for 1977. Can Dis W Rep 1978;3:48-9.
5. Hauschild A, Gauvreau L, Bowmer EJ. Botulism in Canada – Summary for 1978. Can Dis W Rep 1979;5:89-90.
6. Hauschild A, Gauvreau L. Botulism in Canada – Summary for 1979-80. Can Dis W Rep 1981;7:114-5.
7. Hauschild A. Botulism in Canada – Summary for 1981. Can Dis W Rep 1982;8:33-4.
8. Hauschild A. Botulism in Canada – Summary for 1982. Can Dis W Rep 1983;9:53-4.
9. Hauschild A. Botulism in Canada – Summary for 1983. Can Dis W Rep 1984;10:85-7.
10. Hauschild A. Botulism in Canada – Summary for 1984. CMAJ 1985;132:1402.
11. Hauschild A. Botulism in Canada – Summary for 1985. Can Dis W Rep 1986;12:53-4.
12. Hauschild A, Gauvreau L, Black WA. Botulism in Canada – Summary for 1986. Can Dis W Rep 1987;13:47.
13. Hauschild A, Dodds K, Gauvreau L, Smith JA. Botulism in Canada – Summary for 1987. Can Dis W Rep 1988;14:41-2.
14. Dodds K, Davidson C, Proulx JF, Ashton E. Botulism in Canada – Summary for 1989. Can Dis W Rep 1990;16:89-90.
15. Austin J, Dodds K. Botulism in Canada – Summary for 1995. Can Commun Dis Rep 1996;22:182-3.
16. Austin J, Blanchfield B, Proulx JF, Ashton E. Botulism in Canada – Summary for 1996. Can Commun Dis Rep 1997;23:132.
17. Austin J, Blanchfield B, Ashton E, et al. Botulism in Canada – Summary for 1997. Can Commun Dis Rep 1999;25:121-2.
18. Hatheway CL. Botulism. In: Balows A, Hausler WJ, Lennette EH, eds. Laboratory Diagnosis of Infectious Diseases. New York: Springer-Verlag, 1988:111-33.
19. Kautter DA, Solomon HM, Rhodehamel EJ. *Clostridium botulinum*. In: US Food and Drug Administration. Bacteriological Analytical Manual of the Division of Microbiology, Center for Food Safety and Applied Nutrition, US Food and Drug Administration. 7th edn. Arlington: Association of Official Analytical Chemists, 1993:215-25.
20. Centers for Disease Control. Botulism in the United States, 1899 – 1977: Handbook for Epidemiologists, Clinicians, and Laboratory Workers. Atlanta: Centers for Disease Control, 1979:1-41.
21. St Louis ME. Botulism. In: Evans AS, Brachman P, eds. Bacterial Infections of Humans: Epidemiology and Control. 2nd edn. New York: Plenum Publishing, 1991:115-31.
22. Dodds KL, Austin JW. *Clostridium botulinum*. In: Doyle MP, Beuchat LR, Montville TJ, eds. Food Microbiology. Fundamentals and Frontiers. Washington: ASM Press, 1997;288-304.
23. Smith LD. The occurrence of *Clostridium botulinum* and *Clostridium tetani* in the soil of the United States. Health Lab Sci 1978;15:74-80.
24. Smith LD, Sugiyama H. The natural occurrence of *Clostridium botulinum*. In: Balows A, ed. Botulism: The Organism, Its Toxin, the Disease. Springfield: Charles C Thomas, 1988:11-21.
25. Sugiyama H, Woodburn M, Yang KH, Movroydis C. Production of botulinum toxin in inoculated pack studies of foil-wrapped baked potatoes. Journal of Food Protection 1981;44:896-8.
26. Tanaka N, Traisman E, Platinga P, et al. Evaluation of factors involved in antibotulinal properties of pasteurized processed cheese spreads. J Food Prot 1986;49:526-31.
27. Donadio JA, Gangarosa EJ, Faich GA. Diagnosis and treatment of botulism. J Infect Dis 1971;124:108-12.
28. Tacket CO, Shandera WX, Mann JM, Hargrett NT, Blake PA. Equine antitoxin use and other factors that predict outcome in type A foodborne botulism. Am J Med 1984;76:794-8.